Amendments to the Specification:

Please amend the specification as follows:

Please replace the sequence listing filed on September 7, 2001 with the new sequence listing filed concurrently herewith. Please insert the new sequence listing filed following the abstract, and renumber pages 1-9 of the Sequence Listing as pages 47-55. The content of the attached paper copy and the attached computer readable copy of the Sequence Listing are the same.

Please replace paragraph number [0020] with the following rewritten paragraph:

[0020] In a composition of matter aspect, the present invention relates to substantially to a protein comprising a receptor antagonizing domain and a positive immunomodulator domain. The invention further provides that the receptor antagonizing domain can be an apoptosis-promoting domain, while the positive immunomodulator domain can be an interleukin. The receptor antagonizing domain also can be the amino acid sequence ~~SEQ ID NO: 1~~ SEQ ID NO: 34 or conservative variants thereof.

Please replace paragraph number [0026] with the following rewritten paragraph:

[0026] Fig. 5 is a schematic representation of cloning and construction of the expression plasmid of ~~pUCIG-MT-hPRL-IL-2~~ pUCIG-MT-hPRLA-IL-2 fusion protein cDNA.

Please replace paragraph number [0042] with the following rewritten paragraph:

JDK 12/13

[0042] In one preferred embodiment, shown in ~~SEQ ID NO: 1~~ SEQ ID NO: 34, this structural deficiency is a substitution of Gly to Arg at a position corresponding to 129 in hPRL (denoted as hPRL-G129R). Figures 3 and 4, as well as the cell-based assays presented in Examples 4, 5 and 6 demonstrate that this mutated hPRL acts as a true hPRLR antagonist. Accordingly, a receptor-antagonizing domain such as hPRL-G129R can serve as a therapeutic medicament for treating certain types of cancer.